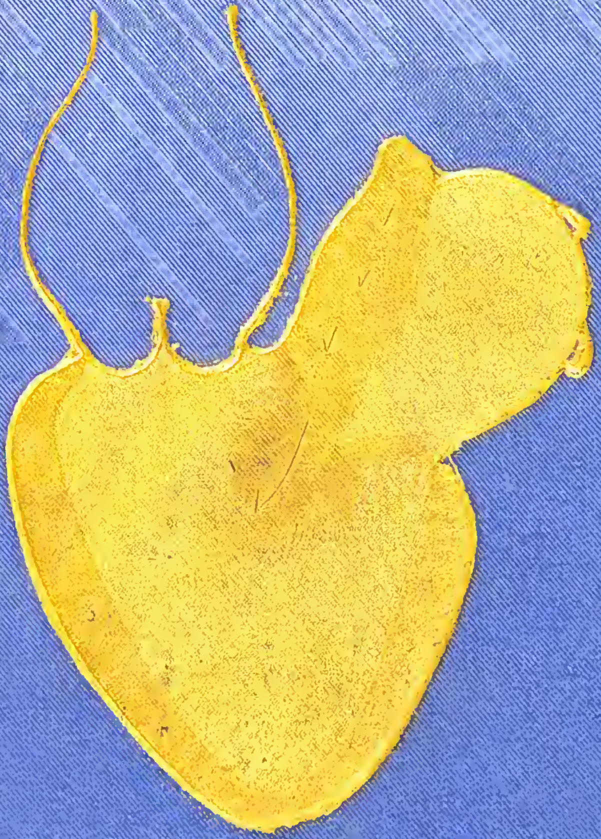
PHYSIOLOGY

OF

THE VASCULAR SYSTEM



MAYO COLLIER

From the Author

ON

THE PHYSIOLOGY OF THE VASCULAR SYSTEM.

THREE LECTURES

DELIVERED AT THE ROYAL COLLEGE OF SURGEONS.

BY

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LECTURES ON THE PHYSIOLOGY OF THE VASCULAR SYSTEM.

LECTURE I.

MR. PRESIDENT and Gentlemen,—I am fully sensible of the honour you have done me in electing me to deliver these lectures. My only fear is that I shall fail, and fail hopelessly, in placing anything before you worthy of the occasion. I am not a physiologist. I am not even a .teacher of physiology. I can only lay claim to be an ordinary observer, but as such I have endeavoured to pay some considerable attention to the phenomena of the circulation. As a teacher of anatomy I am constantly demonstrating the cavities and valves of the heart, and I have found it impossible to dissociate the physiology from the anatomy of these parts. Indeed, the student will not rest content with the dry anatomical facts.

Constantly I am asked the question, when speaking of the pouches at the root of the aorta and pulmonary artery, What is the use of them ? or when demonstrating the parallel fibres in the auricular appendages, What are they for ? In order to make a demonstration on the heart interesting, or at least complete, it is necessary for the teacher of anatomy to trench on the province of the physiologist, to describe the course of the blood through the cavities of the

heart and the mechanism and action of the valves. From this you will see that at certain points anatomy and physiology are inseparable; in fact, physiology is the interpretation of anatomy. At certain points, then, a teacher of anatomy must be ready and prepared to discuss and interpret the arrangement of parts.

Now, sir, it was this necessity on my part, when teaching the anatomy of the heart and great vessels, to at the same time explain the working of the valves and the general mechanism of the parts, and my absolute inability to accept the account of the same as given in the text-books, that led me to pay a little more attention to the subject than I should have otherwise done, and to endeavour to think the matter out for myself. This, then, is my excuse for dealing with a subject that may otherwise be considered outside my province.

In 1628 William Harvey wrote, "The blood within the vessels is in a state of continual motion, being carried forward from the ventricles by the large arteries (aorta and pulmonary) and their branches to a system of capillary vessels, from which again, it passes into the veins that end in the atria of the auricles." If I were to say that this statement contains all that is actually known, that is to say known to a certainty, not a matter of opinion, of the phenomena of the circulation in 1889, you would probably disagree with me. True, to-day we have the sphygmograph, and can take a tracing of the movements of the arteries; but when we have got a tracing can we interpret it? do we know what it means? True, we have the cardiograph, and can take a tracing of the movements of the heart, or fancy we can; and what next? can we interpret it? True, we have the stethoscope, and can listen to the sounds of the heart; but can we interpret what we hear? Do we know, are we all agreed as to the causes of those sounds? The answer to all these questions must be, No. We think, but we do

not know. Our knowledge on the subject is still a matter of opinion.

It was no matter of opinion of Harvey's that the blood flowed in a particular direction. It is a truth, an absolute fact, an incontrovertible statement, capable of easy demonstration and of absolute proof. If, then, I were to contend that the additions to our store of absolute truths or facts in connection with the vascular system since 1628 were very meagre, I do not think I should be far from the mark.

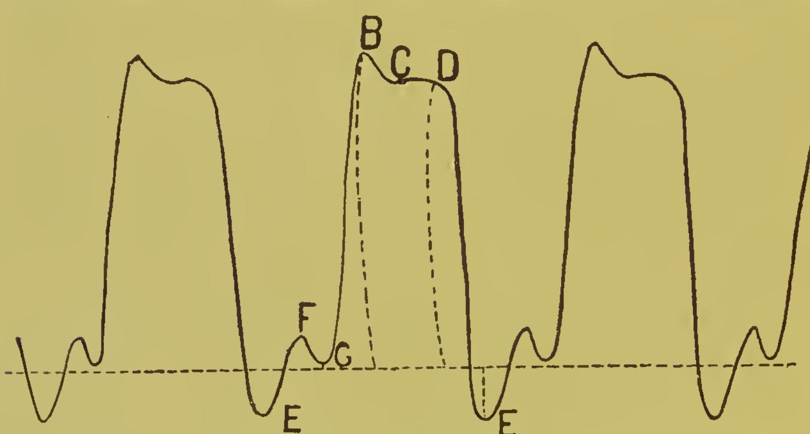
Considering, then, how great is the importance of a true understanding of the phenomena of the circulation, how vast is our want of knowledge on the subject, and how great is the extent of the region that requires investigation, however small the work done, and however humble the contribution to the store of our knowledge, provided it is real knowledge, it cannot fail to be acceptable to us.

We are all agreed that the blood moves in one constant direction, from the ventricles to the arteries, from the arteries to the capillaries, then to the veins, and back to the auricles. The cause of this movement is equally clear and certain, namely, the difference in pressure that exists between the aorta and pulmonary artery on the one hand, and the vena cava and four pulmonary veins on the other. By direct experiment we have found out that the difference in pressure is greatest between the aorta and vena cava and between the pulmonary arteries and veins. The pressure in the aorta is stated to range between 200 and 300 mm. of mercury, and in the venæ cavæ from -20 mm. during diastole to perhaps $+20$ mm. during systole of the ventricle. The maintenance of the difference between the 200 to 300 mm. of mercury in the aorta and the $+20$ to -20 mm. in the venæ cavæ is the work of the ventricle.

So far we are all agreed; it is when we come to consider the

method of contraction and dilation of the ventricle and the attendant phenomena that we are not of one mind.

We have before us a ventricle, composed of several hollow cones of muscular tissue. We know that muscular tissue is endowed with a perfect elasticity, and we might infer from this, if not otherwise capable of proof, that the ventricle, when its cavity was obliterated or lessened, would return *per se* to its original shape, the cause of the alteration of its shape being



INTRA VENTRICULAR PRESSURE CURVE.

removed. This inference is supported and converted into fact by the results of direct observations on the empty and full hearts of animals. We are all familiar with the experiments and tracings of Marey and the observations of Magini, but it is to the experiments and tracings of Mr. H. D. Rolleston, of St. John's College, Cambridge, that I would specially draw your attention.

These experiments have been so carefully planned and admirably carried out that I venture to regard the main results as conclusive. I say the main results, because the correct interpretation of these tracings is beset with difficulties, and it is the reverse of

wisdom to attempt to explain one difficulty by creating others. However, the main results, I think, we can accept without reserve. I have repeated these experiments, and without pretending for a moment to have carried them out with the exactitude and care of Rolleston and others, still my experiments fully satisfied me that from the commencement of diastole the pressure in the ventricle suddenly and rapidly fell to a point in some cases considerably below the atmospheric pressure. Associated with the fall there was a distinct flattening of the great veins and auricle. The fall is indicated in the chart (page 4) by the lines B C D E. The portion below the horizontal line marks the fall below atmospheric pressure. There is no notch indicating increase of pressure due to an auricular systole; the fall is rapid, continuous, and regular. The only possible interpretation of this is that the ventricle exerts a powerful aspirating effect on the auricle and great veins.

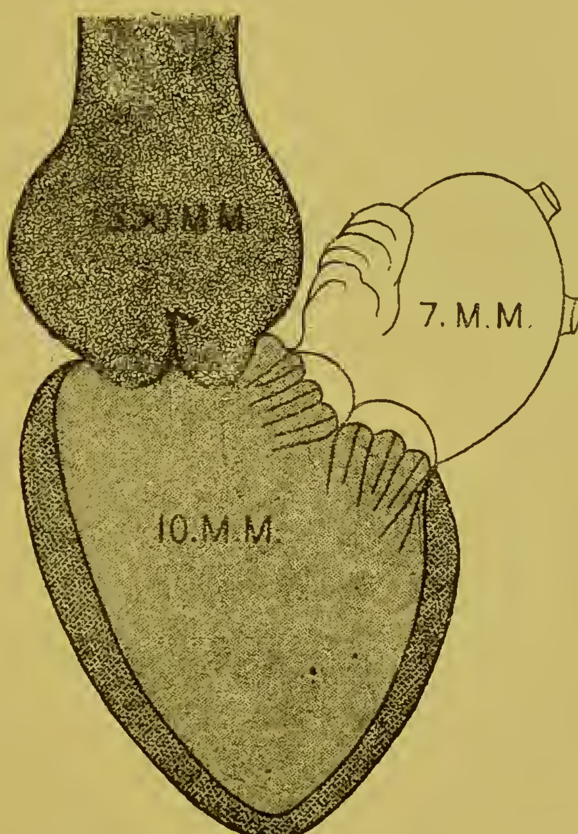
On the settlement of the question of the active dilatation of the ventricle, and the powerful aspiration of the auricle and great veins, a great deal depends, for, as I shall attempt to prove later on, it is impossible to see how the ventricle can become rapidly filled without this. I hold that it is at the very foundation of the whole question. I am not going to subject you to many quotations during these lectures, but so important do I conceive the settlement of this question to be that I shall quote from Mr. Rolleston's paper a few lines bearing directly on the subject. Under the heading "Diastolic Fall of Intra-ventricular Pressure" he says, "The manometric curve of the intra-ventricular pressure falls usually fairly sharply from the point D, afterwards descending more slowly. In certain cases it falls below the atmospheric pressure, as is seen in curves i, ii, iii, iv. The negative pressure occurring during the expansion of the muscular wall of the ventricles is met with both in the right and left ventricular cavities. With

regard, first of all, to the value of the negative pressure, this is found to vary in different animals and in the case of the same heart under different conditions. On measuring out my curves, I find that some show a negative pressure at the point E equal to 20 mm., whilst others show varying slighter negative pressures. In the case of some hearts my curves show no fall of pressure below that of the atmosphere. These latter cases, however, are less frequently met with than those showing a more or less well-marked negative wave at the end of the descent."

Speaking, farther on, of the meaning of the negative fall below the atmospheric pressure, he says, "The explanation which best accords with the facts before me is that the elastic expansion of the ventricle lasts longer than it appears to have been assumed by Goltz and Gaule, and that it may continue after the blood in the auricle, at the moment of cessation of the ventricular systole, has entered the ventricle."

"This theory would assume that the negative pressure would appear also in the auricle, and that it is due, in fact, to the quantity of blood in the auricle and terminal trunk of the pulmonary veins not being sufficient to distend the left ventricle to the point at which its suction action ceases. The absence of any sub-atmospheric pressure in the same auricle at different periods of the experiment could thus be readily explained by the variations in the quantity of blood available to fill up the *potential vacuum* caused by the elasticity of the expanding heart-wall. In support of this view I may say that sub-atmospheric pressures are met with in the auricle, as I have found in at least one well-marked case while recording the pressure within the left auricle." Such is Mr. Rolleston's view. When speaking of the method of closure of the auriculo-ventricular valves I shall offer another explanation of this negative pressure, a direct outcome of the approximation of the parachutes.

With most of the first quotation I entirely agree, and I put it to you that with this clear, unmistakable proof of a rapid, continuous, and regular fall of endo-ventricular pressure without a single notch or rise, what other possible interpretation is there but that it is due



Ventricle full. Valves closed previous to Systole.

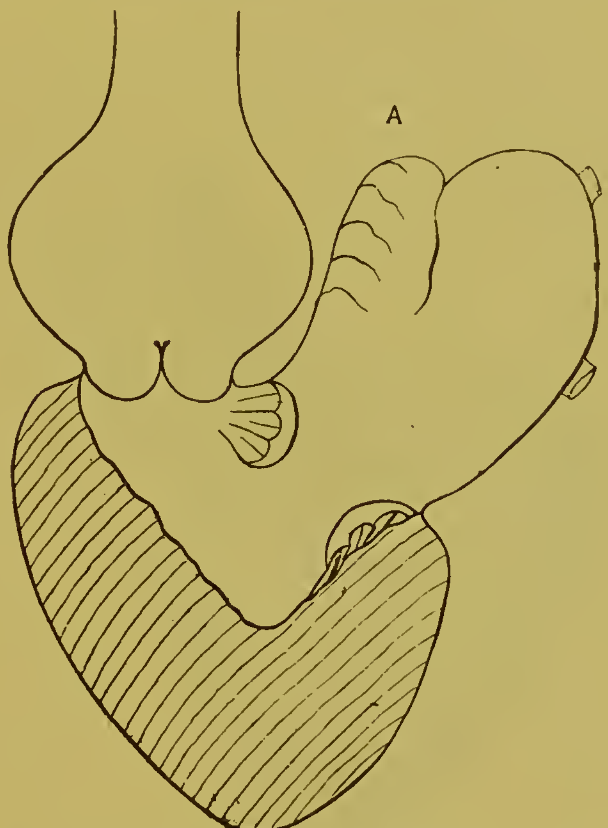
to the active dilatation of the ventricle and its aspirating effect on the auricle and great veins?

I think we may take it, then, as an established fact that the ventricle is capable of and does actually expand *per se*, so as to suck blood with considerable force from the great veins and

auricles. This fact being established, the necessity for a vigorous contraction of the auricle ceases, and the absence of valves at the root of the great veins and in the liver is explained.

Leaving, for the present, the discussion as to whether the auricle as a whole contracts, and what part the auricular appendices play, to a later period, we now contemplate the ventricle distended with blood, ready to propel its contents into the aorta or pulmonary artery, as the case may be. Before any blood can enter the aorta from the ventricle the pressure in the latter must be changed from – 28 mm., or perhaps + 2 mm., to over 250 mm., 300 mm., or perhaps 350 mm., and before the ventricle can completely empty itself its contents must be exposed to a very much greater pressure even than this. The effect of contraction of the ventricle, if the passage into the auricle be not shut off by closure of the tricuspid and mitral valves, would be simply to force blood back into the auricle without increasing materially the tension in the ventricle. The argument that the ventricle contracts so suddenly that the valves are floated up by the back rush of blood into the auricle would imply regurgitation into the auricle as a normal state of things, and this slap-bang action would subject the valves to the constant risk of rupture. Moreover, any sensible regurgitation into the auricle would cause a pulsation and temporary arrest of flow in all the larger veins leading into the auricle. I submit that no such thing takes place, nor can I accept the explanation that a reflex current curls round the outside of the valves, and floats them up in opposition to the inflowing blood from the auricle. The inflowing blood from the auricle must exert a considerable pressure on the auricular surface of the valves, with the result of separating the valves and pressing them against the wall of the ventricle. Some active agent must be present whereby the valves are drawn away from the ventricular wall and approximated in the face of and in opposition to the inflowing blood from the auricle.

It seems to me to be absolutely requisite that the valves should close to before the tension in the ventricle can commence to rise. Therefore I would suggest that the first act of the ventricle is to bring together the tricuspid and mitral valves. It appears to me



Ventricle aspirating Auricle.
Commencing Diastole of Ventricle. A, Auricular Appendix.
Valves pressed back against Ventricular Wall by overflowing Blood.

to be not inconsistent with what we see in other parts of the economy, namely, that involuntary muscle is capable of slow, prolonged, and vigorous contraction, commencing in one part and gathering force as the wave of contraction proceeds by involving

more fibres, the effort of contraction suddenly relaxing. I would suggest that the contraction of the ventricle commences in the musculi papillares ; that the musculi papillares begin to contract from the commencement of the diastole of the ventricle, and so

Ventricle nearly full. Appendix closing Valves.

bring the valves together. This method of closure will explain the cause of the sub-atmospheric pressure, because the approximation of the parachute makes it more difficult for the blood to enter from the auricle towards the end of the diastole. In fact, the entrance of the blood from the auricle has to be assisted by

the contraction of the auricular appendix, as I shall point out later on. Thus it would appear that the valves are brought into apposition before the ventricle is quite full; that as soon as the ventricle has ceased to dilate, and the tension in the ventricle commences to rise, the valves become closely approximated and remain efficient, (diagram, page 10).

I am perfectly well aware that this explanation of the action of the ventricle involves the rejection of the apparently well-accepted statement that the first sound of the heart is in part due to the sudden closure and vibration of these curtains. I am not alone in holding this view. I could mention some names of great weight whose decided opinion is that the auriculo-ventricular valves are closed before the tension commences to rise in the ventricle. I maintain that any other method of closure, either by back wash, or reflex current, or what not, implies, of necessity, regurgitation into the auricle as a normal state of things, and there is not the smallest particle of evidence that anything of the kind takes place.

To repeat more exactly, then, I would maintain that the ventricle aspirates the auricle; that the musculi papillares commence to contract from the very commencement of the diastole; that thereby the parachutes are drawn away from the ventricular wall and approximated, the blood flowing in the while from the auricle; that the more distended the ventricle becomes the more unfolded and distended are the parachutes.

Most of the above remarks were committed to paper and published some eighteen months ago. Since then I have had the pleasure of reading this paper by Mr. Rolleston, and have the gratification to find his conclusions, although arrived at by investigating the subject from a different point of view, in most points corroborate and support mine.

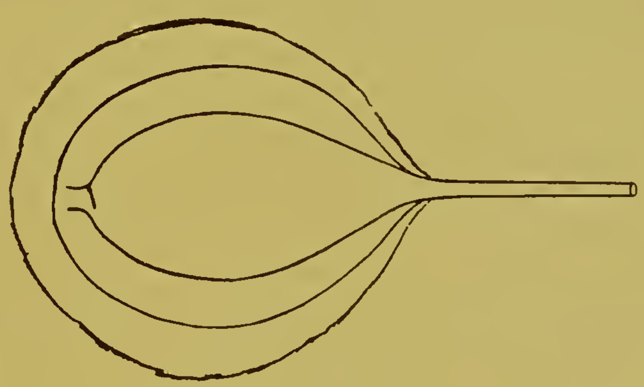
At the end of his paper, under the heading "Conclusions,"

appears the following paragraph: "As to the points which correspond in the intra-ventricular pressure tracings to the time at which the auriculo-ventricular valves are closed and the sigmoid valves are open and shut, my observations lead me to conclusions which are different from those arrived at by Marey. I am forced to conclude that these valves must be shut before any considerable rise of pressure can take place within the ventricle above that which results from the auricular systole. The closure of the auriculo-ventricular valves which must take place at some point in the lower third of the rise, G B in my figures, does not produce any notch or wave in the tracing, which must of necessity be the case if they shut to suddenly." Moreover, the impact of the valves on the auricular contents would produce a rise of pressure in the auricle, which should and would be indicated by the manometer. Now, no such indication is given. The opening into the auricles being closed by the efficient approximation of the valves, the contents of the ventricle is subjected to a gradually increasing pressure until the pressure or tension within the ventricle is changed from – mm. to + 250 mm., or equal to the tension in the aorta. At this precise moment the pressure on both sides of the valves is equal. When the pressure within the ventricle exceeds the pressure within the aorta the valves will commence to separate and blood to enter.

I would here remark that the act of the ventricle in discharging its contents into the aorta is a complicated one. It is not simply discharging itself into an empty receptacle, but into an elastic bag already over-distended and exerting considerable pressure on its contents. A great many questions are here involved, the most important being the method of opening and closing of the semilunar valves, the position of this extra quantity of blood in the aorta, the effect produced on the aorta, and the general effect on the circulation.

A few words in reference to the laws of flow in rigid and elastic tubes will here not be out of place.

In rigid tubes full of fluid under a certain pressure if a quantity of fluid be forced in at one end the same quantity will be forced out, particle for particle, in the same time at the other end. For the sake of illustration we may consider the tube as consisting of polished steel, and the contents as small round pieces of ice. The same number of pieces of ice that enter at one end would leave in the same time from the other end, particle for particle.



Elastic Ball with Rigid Tube.

If now an elastic ball be attached to one end of the rigid tube distended with ice a continuous flow from the distal end of the tube will be the result, lasting till the ball has returned to its normal undistended capacity. If while the ball is distended and driving its contents through the rigid tube more particles of ice be introduced into the elastic ball the effect will be seen by (1) a distension of the ball, and (2) by an increase in the rapidity of flow from the open end of the tube.

I shall presently point out that at each contraction of the

ventricle, amongst other effects, there is undoubtedly a distension of the first part of the aorta, and an acceleration of flow from the cut end of an artery synchronous with the action of the ventricle. So far, then, the aorta and arteries resemble a rigid tube with an elastic ball, distended as in our illustration. In an elastic tube immediately after the forcing in of a quantity of fluid only part flows out, the rest continuing to flow after the propelling force has ceased to act. When an uninterrupted uniform current flows through an elastic tube it follows the same laws as if the tube had rigid walls. If the propelling power increases or diminishes the elastic tube becomes larger or narrower, and it behaves, as far as the movement of the fluid is concerned, as a wider or narrower rigid tube. If, however, more fluid be forced or jerked in an elastic tube interruptedly the first part of the tube dilates suddenly, corresponding to the amount of the fluid propelled into it. The greater the rapidity of the injection the more sudden the dilatation of the first part of the elastic tube, and the more sudden the dilatation the more sudden the recoil.

We have seen that when the fluid is injected into an elastic ball in connection with a rigid tube the recoil of the elastic ball will have the result of, and will be entirely expended in, accelerating the flow, because the recoil is incapable of reacting on the rigid tube. It is otherwise when the tube is elastic. The sudden dilatation of the first part of the tube is followed by an equally sudden recoil. There is nothing whatever to prevent the recoil affecting a segment of the elastic tube next to it, and suddenly dilating it, and so on to the end of the tube, according to the rapidity of the initial injection.

Now, I maintain that from our experiments with elastic and rigid tubes and a combination of the two we gather three points that will assist us when examining the injection of the aorta by the ventricle :—

1. That the injection distends only the first part of the elastic tube (immediately).

2. That the flow from the cut end of the elastic tube is increased during the injection.

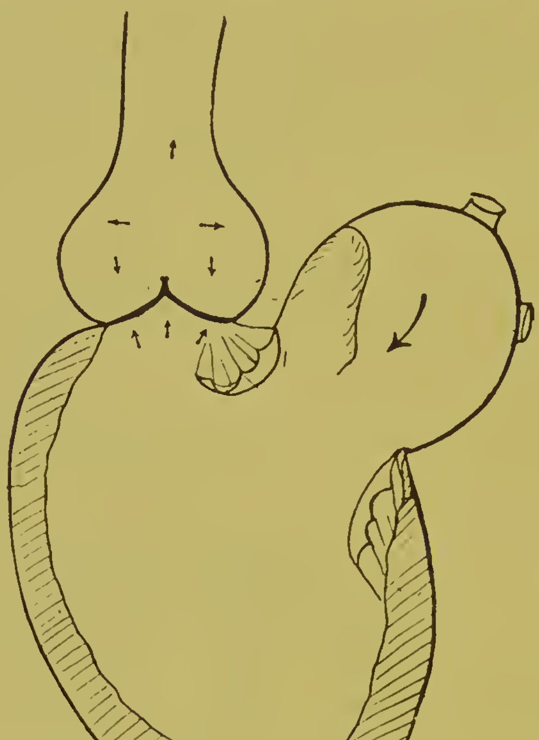
3. That the recoil of the dilated first portion of the tube rapidly affects a portion of the tube nearest to it, and then another portion, and so on ; or, in other words, the sudden increase of tension is quickly distributed from one end of the tube to the other. Or, in other words, the first part of the tube being distended suddenly by the pump, distends a second portion, and maintains an increased tension of its own, which increased tension subsides to the original tension more or less quickly, and so on, segment after segment. The second portion so distended recoils and parts with some of its increased tension, to dilate and increase the tension of a third part, maintaining an increase of tension of its own, and so on, according to the amount of the original distension. So much we can learn from experiments on rigid and elastic tubes.

I would here lay special stress on the point that when fluid is injected suddenly by jerks, that is to say interruptedly, into a system of elastic tubes with modified resistance, it is the first part only of the elastic tube that is dilated. This can at all times be directly proved :—

1. On the living aortæ of animals.
2. By observing the result of injections on elastic tubes.
3. By what I may call the mathematical proof.

At the Brown Institute, in conjunction with Professor Horsley, I opened the thorax of large dogs, and placed fine aluminium calipers on the aorta and measured the expansion. The expansion was considerable, and could to my mind quite account for all the blood delivered by the ventricle. I repeated this experiment on dogs of all sizes, with the same result. No absolutely correct or mathe-

matical results could be obtained, from the impossibility of eliminating the errors due to the movements of the diaphragm and lungs. One thing was absolutely certain and constant, that at each contraction of the ventricle the aorta nearest the heart was consider-



Ventricle aspirating Auricle.
Arrows indicating direction of pressure in Aorta.

ably distended, as evidenced by the dilatation of the points of the calipers, distended, as I presume, sufficiently to account for the whole of the extra blood delivered into the aorta by the ventricle.

It is a matter of common observation, and may be illustrated by

the simplest experiment, that if water be forced into an elastic tube with modified resistance at end, interruptedly, the first part of the tube dilates suddenly, the distension being quickly distributed through the rest of the tube.* This simple tube and bag will illustrate my meaning. The tube is made thicker here, to resist the distension of the first portion.

The mathematical proof, to my mind, is complete and unanswerable. The energy of the ventricle will be expended partly in raising the semilunar valves, and partly in thrusting into the already over-full aorta an additional quantity of blood. The unit of pressure on the valves on the interior of the aorta and on the cross-section of the aortic contents at a certain distance from the valve will be the same, because fluid pressure exerts itself equally in all directions. Now, as the area of the aorta is many times greater than the area of the cross-section (diagram, page 16), the greater part of the energy of the ventricle will be expended in dilating the first portion of the artery, and only a comparatively small amount in driving forward the main column of blood, *i.e.*, accelerating the flow.

So important do I consider the establishment of the fact that it is the first part only of the aorta that is dilated by the contraction of the ventricle, and that this dilation passes in regular wave from one end of the arterial system to the other, that I had adduced the foregoing arguments in proof, any one of which, to my mind, is sufficient.

On the establishment of this fact depends the proper understanding of the mechanism of the pulse and the mode of closure of the semilunar valves.

As a further illustration, consider the explosion in an ordinary fowling-piece. The force of the explosion is expended equally in all directions—forwards, backwards, and sideways. The action

* Here was shown a Politzer's bag, the tube leading from the bag being made thicker to resist the distension from the sudden contraction of the bag.

and reaction are equal and opposite at all points of the compass. Why is it necessary to have the gun for several inches thicker at the breech than elsewhere? Because the amount of pressure being equal in all directions, and the area of the walls of the breech being many times greater than the cross-section of the barrel, the chief effect of the explosion would be expended in dilating the breech, and only a relatively small amount in driving forward the shot. The breech, however, is prevented from dilating by its strength, consequently nearly the whole force is concentrated on the projectile. This homely illustration may possibly serve to make clear my contention that it is the first portion only of the aorta that is dilated as the immediate effect of the ventricular systole.

Before leaving the subject of the laws of flow in rigid and elastic tubes, at the risk of being tedious I should like here, in as few words as possible, to explain how an intermittent injection into elastic tubes becomes converted into a continuous stream. The flow would be intermitting in character in elastic tubes if the time between two successive systoles were longer than the duration of the current necessary for the compensation of the difference of pressure caused by the systole. If the time between two successive systoles be shorter than the time necessary to equilibrate the pressure the current will become continuous, provided the resistance at the periphery of the tube be sufficiently great to bring the elasticity of the tube into action. The more rapidly systole follows systole, the greater the difference of pressure becomes, and the more distended the elastic walls. Although the current thus produced is continuous, a sudden rise of pressure is caused by the forcing in of a mass of blood at every systole, so that with every systole there is a sudden jerk and acceleration of the blood stream.

The idea, then, that the blood flows in a stream directly through

the aorta by the direct propulsion of the ventricle cannot be maintained for a moment; and equally untenable is the statement that the particles of blood composing this stream, obeying the first law of motion, continue to move after the ventricle has discharged itself, and so causing a diminution of pressure near the valves, induce a back current, which closes the semilunar valves.

We are now, then, in a position to follow the blood through the semilunar valves into the aorta, and note the result.

The observation "that when in a closed channel a rapid current suddenly ceases a negative pressure makes its appearance in the rear of the fluid and sets up a reflex current" is perfectly true, but I fail to see in what possible way it has the slightest bearing on the injection of the aorta by the ventricle. The observation applies only to a rapid current passing through rigid tubes with a free exit. No reflex current is possible in a tense elastic tube under very great pressure.

We previously digressed at the point where the tension in the ventricle had risen after the closure of the auriculo-ventricular opening from -2 mm. to $+200$ mm., that is to say to a part where the tension in the ventricle and aorta was equal. At this period obviously the pressure on both sides of the semilunar valves is the same. At the moment when the pressure in the ventricle exceeds the pressure in the aorta the valves will move up, separate at their centres, and blood will enter. As blood is squeezed into the aorta from the ventricle the tension in the aorta increases and consequently reacts and opposes the further action of the ventricle, necessitating and calling forth an increase of energy from that structure. The arrangement of the fibres of the heart in distinct cones provides admirably for this, allowing a diminution of the cavity of the ventricle, and at the same time keeping in reserve several cones as emergency demands. The ventricle, then, from the moment it commences to inject blood into the aorta, has to

exert a gradually increasing force, the tension being greatest at the end, and least at the beginning of the systole, both in the aorta and ventricle. From this we can make a most important note, namely, that the blood towards the end of the systole enters the aorta under very great pressure, and consequently greatly diminished momentum; and further, that the rate of injection by the ventricle gradually decreases from the beginning to the end of the systole. At the end of the systole, when the pressure above and below the valves is again equal, the valves are just closed, and the last drop of blood that enters must occupy the position of the corpora arantii. While the pressure in the ventricle exceeds the pressure in the aorta blood will enter from one into the other. Directly the pressure in the ventricle begins to relax the pressure on the upper surfaces of the semilunar valves will close them down, and at the precise moment when the pressure on both sides is equal no more blood will enter. At the precise moment when the pressure in the aorta is greater than that in the ventricle by ever so small in amount the valves will be closed to completely.

The next point to investigate is the position in the aorta occupied by the contents of the ventricle. For the sake of illustration we may look upon the aorta as an elastic bag distended with little solid particles, say peas. Now, if one pea be thrust into the bag it will slightly further distend it, and rest immediately above the corpora arentii, exerting a pressure in every direction—forwards backwards, and sideways. The same argument will apply if fifty peas are thrust in; they will occupy the neighbourhood of the centre of the upper surface of the valve and the surrounding space. Extending our illustration, the blood enteiing from the ventricle will occupy the centre of the lumen of the first few inches of the aorta, thrusting the previous contents forwards, so accelerating the flow through the arteries generally, backwards

exerting an increased pressure on the semilunar valve, and sideways tending to greatly distend the first part of the aorta It is clear, then, that blood traverses the arterial system in the order in which it enters the aorta from the ventricle.

Position of Ventricular Contents in Aorta.

To repeat more exactly, the following is my contention so far : That the blood in the ventricle is sucked in from the great veins and auricles by the aspirating power of the ventricle. That the musculi papillares commence to contract and approximate the

valves before the ventricle is quite full. That by the time the ventricle is fully distended these valves are approximated. That as soon as the tension in the ventricle begins to rise they are closely approximated and rendered efficient. The tension in the ventricle now rapidly rises, more and more of the heart substance being called into action. As soon as the tension in the ventricle exceeds that in the aorta blood commences to enter, the valves at the same time separating at their centres. The direct effect of the blood entering the aorta from the ventricle is an increase of tension and distension of the first part of the aorta, an increase in the rapidity of flow in the small arteries, and an increase of resistance to the ventricle. Towards the end of the systole the valves are nearly closed, and the last few drops of blood enter the aorta under a maximum of pressure and with small momentum, and lie above the corpora arantii. The blood thrust in by the ventricle occupies the centre of the lumen of the first few inches of the aorta.

LECTURE II.

MR. PRESIDENT and Gentlemen,—In my last Lecture I endeavoured to lay before you what I conceived to be a more correct account of the contraction of the ventricle, and its effect upon the aorta, valves, and general circulation. I was the more concerned in establishing three points :—

1. The powerful aspiration by the ventricle of the auricle and great veins.

2. That the chief part of the energy of the ventricle was expended in distending the first part, and the first part only, of the aorta.

3. That the contents of the ventricle were deposited in and distended this first part of the tube.

To-day I propose to direct your attention to the three small pouches at the root of the aorta and pulmonary arteries, and endeavour to prove to you that they are not only not semi-pathological formations, as stated by most authors, but, on the contrary that they are absolutely essential to the mechanism of the ventricles; in fact, without these the constant efficiency of the heart as a pump would be seriously endangered.

The auricular appendices, hitherto regarded, or rather disregarded, I shall endeavour to show are very important agents in the closure of the tricuspid and mitral valves. My views on the functions of these parts were embodied in a paper placed before the Royal Society some eighteen months ago, but I regret to say subsequently deposited in the limbo of the Proceedings without comment and without criticism. With your permission I shall

embrace this opportunity of again submitting them to your notice, feeling sure that in submitting my views to this the highest tribunal the chaff will be quickly separated from the wheat, and after that process if there is any residuum, I offer it for your acceptance.

The sinuses of Valsalva are three pouches situated at the root of the pulmonary artery and aorta, opposite the semilunar valves, one for each. They are permanent or structural dilatations in the walls of these vessels, and as such are easily demonstrated on the undistended and empty tubes. These pouches are constant features of all mammalian and avian hearts. Up till quite recently they were simply described by anatomists, and no particular part was assigned to them in the mechanism of the circulation.

Recent authors, asserting the existence of a reflex current in the aorta, have built upon this assumption, and used it as an explanation of the existence of these pouches. The argument is this, that a reflex current exists in the aorta; that this reflex current strikes the upper surface of the semilunar valves, and from thence is reflected on to the wall of the artery opposite these valves; that by the gradual effect of this reflex current these pouches are formed. In support of this argument the statement is adduced that these pouches do not appear till late in fœtal life, and from this it is argued that their late appearance is due to the gradual effect of the back or reflex current during intra-uterine life.

Let us follow this argument to its legitimate conclusion, allowing, for mere purposes of argument, that such a state of things as a reflex current does actually exist. Surely as the heart becomes stronger this reflex current will increase in force, and consequently the effect on the wall of the artery will increase *pari passu.* The gradual dilatation will not cease, but may reasonably be expected to increase with advancing years, and we might legiti-

mately expect to find the sinuses of Valsalva assuming proportions frightful to contemplate. As a matter of fact the sinuses of Valsalva appear very early in fœtal life, assuming commensurate proportions with the growth of the heart. It is usual to find that the pouches are not equally developed ; one is usually in a rudimentary state till quite late.

Sinuses of Valsalva. Valves thrown up.
Dotted Line indicating Cylindrical Aorta. Valves in close apposition to same.

I think, then, I have disposed of the explanation as at present given in recent works of physiology of the quasi-pathological formation of these pouches. Under normal circumstances the semilunar valves can never line the sinuses, however much they are pressed up or inverted by the contents of the ventricle because the capacity of the sinus or area of the sinus is much

greater than the capacity or area of the inverted valves (diagram, page 25). At all times, under normal circumstances, blood in considerable quantity, exerting a considerable downward pressure on the valves, will exist in these sinuses.

Let us now contemplate the result of the non-existence of these pouches; let us for a moment assume that the root of the aorta is quite cylindrical. Under these circumstances a sudden and violent contraction of the ventricle could so displace these valves as to approach them closely to the wall of the aorta, with the result of fixing them in this abnormal position (diagram, p. 25). Once let

Elastic Bag illustrating Initial distension of Aorta by Ventricle.
V, Ventricle; A, Aorta.

them line the arterial wall, and the whole pressure of the aorta would be thrown upon the under instead of the upper surfaces, so the more tending to retain them in their abnormal positions, and leaving the ventricle to support the whole pressure of the aortic contents. In the case of at least two of these pouches the suction of the coronary arteries might retain two of the valves and prevent any further supply of blood to the heart.

I would here offer as a mere suggestion that in some cases of sudden death from emotional causes, in weakly subjects where the tension is low, and where no post-mortem structural change is found in the heart, that the state of things I

have described above as being impossible under normal circumstances does actually take place.

I submit to you that the sinuses of Valsalva prevent the semilunar valves from ever, under normal circumstances during life, being sufficiently inverted as to line these pouches, and thereby constantly maintaining a state of things by which under all circumstances the valves shall come together subsequent to the injection by the ventricle. Such, then, being the case, they are absolutely essential to the efficient action of the valves.

The auricular appendices, as you are aware, are conical

INTRA AURICULAR PRESSURE CURVE.

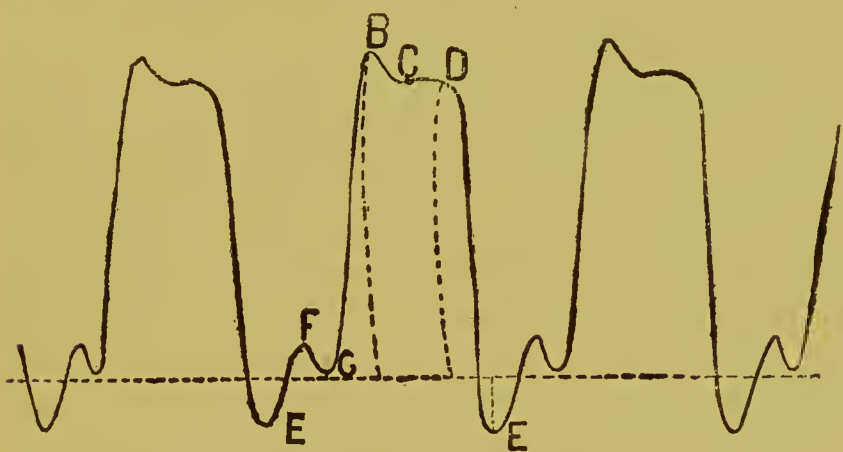
dilatations in the upper and fore part of the auricular chambers. They are situated immediately above each auriculo-ventricular orifice. They are constant features of all mammalian and avian hearts. The auricular appendix is the strongest and thickest part of the auricle. In these structures the fibres are arranged in powerful parallel bundles, which stand out in bold relief on the inner surface, suggesting the name, musculi-pectinati. In the rest of the auricle the fibres are very much more sparse, running in all directions, leaving intervals composed simply of peri- and endo-cardium.

Having grave doubts as to the possibility of the auricle as a whole actively contracting, and still graver apprehensions that if

such were the case the result could only be injurious to the circulation, I instituted a number of experiments on the auricles of dogs. In the first place I failed to observe that the auricle ever did empty itself. During the diastole of the ventricle a distinct flattening of the superior cava and upper part of auricle was noticeable. I could detect no wave of contraction spreading from the great veins towards the auricle. The only portion of the auricle that was observed to contract sensibly and vigorously was the auricular appendix. The contraction immediately preceded the systole of the ventricle, and might be compared to the descent of the hammer on the cap before the explosion in the gun when the heart was beating rapidly. The appendix completely emptied itself, became pale and shrivelled. Next, in order to ascertain the absence or presence of increase of tension in the auricle (such as undoubtedly would be the case if the auricle contracted vigorously as a whole), the interior was placed in communication with the limb of a manometer by introducing a cannula into the cavity of the chamber. No marked rise of the other limb of the manometer could be observed; on the contrary, an oscillation and advance of the mercury on the limb of the manometer connected with the auricle was evident.

I place before you here an endo-auricular pressure tracing after Mr. Rolleston, taken with the most minute precautions, to ensure accuracy, with Professor Roy's manometer. Turning to the tracing marked No. 5 in his work, the author describes it as an auricular wave from cavity of left auricle thorax opened. To take this tracing at all very small resistance had to be employed, and you see it simply represents a slight rise of pressure which takes place in the auricle subsequent to the closure of the parachutes, and due to the blood collecting in the auricle and great veins. Turning to tracing marked "Endo-ventricular Pressure

Curve," the author says, "With regard to the auricular systole, it can be seen in figures 1 and 4 that this event causes no rise of pressure separate from that due to the ventricular systole. This absence of a distinct rise of pressure in the ventricle, resulting from the auricular systole, is one of the most striking features of the curves obtained." And later on he says, "I am therefore obliged to assume that the contraction of the auricle in the dog does not cause any rise on the intra-ventricular pressure curve of a kind similar to that obtained by other observers in the case of a horse."



INTRA VENTRICULAR PRESSURE CURVE.

I agree with the author if he says there is no rise due to a contraction of the auricle as a whole, because I am endeavouring to prove to you that the auricle does not contract as a whole; but in reference to the auricular appendix I say the tracing shows a most distinct elevation at the point F, due to the increase of pressure imparted to the ventricle by the contents of the appendix. A slight fall succeeds this initial rise, because thereby the parachutes are bellied out towards the auricle, and prevent any further entrance of blood from the auricle. The capacity of the

ventricle is thereby slightly increased, and consequently the pressure is slightly lowered. You will naturally ask the question, How is it that the addition of the contents of the appendix affects the valves in preference to the rest of the ventricular wall?

My answer is this: The pressure in the ventricle will be at every point equal. Any addition of pressure will affect every part of the interior equally, but will distend the weakest and thinnest parts most. Now, as the valves form part of the ventricular chamber, they will be distended in preference to the thick muscular wall.

Another question presents itself. All authorities agree (even the advocates of a wave of contraction spreading from the great veins) that the auricle, if it contracts at all, contracts suddenly, and immediately before the ventricle. Some authors assert that the two acts are continuous, and again that the auricular systole merges or passes into the ventricular systole. I ask you, looking at the thinness and constitution of the wall of the auricle apart from other evidence, how is it that a thin-walled chamber like this, is capable of contracting on a mass of blood with lightning rapidity, and driving its contents into an already—mark you—full ventricle. Nobody ever claimed for the auricle that it contracted at the commencement of the diastole of the ventricle. If it were so, one could understand how it might come about that a thin hollow chamber like the auricle could empty itself, but not suddenly and in the face of a full ventricle.

Next, what evidence can we adduce from the records of pathology for and against the theory of an auricular systole apart from that of the appendix?

1. A loud presystolic murmur. I do not admit this as evidence in support of a vigorous contraction of the auricle; in the first place, because the appendix is an all-sufficient cause; secondly,

because the aspiration of the ventricle is an all-sufficient cause; and lastly, but not least, because the auricle has been found so dilated and thinned as to be physically incapable of anything bordering on a vigorous contraction, and yet a loud presystolic or post-diastolic murmur has been evident during life. Dr. Sansom has directed my attention to one case, in his Lettsomian Lectures, where a very loud presystolic murmur, varying with a post-diastolic, existed during life. At the post-mortem the auricle was found to be greatly dilated, and lined to the extent of half an inch with laminated fibrine.

2. Great hypertrophy and thickening of the whole auricle, associated always with dilation, would appear to favour the view of an active contraction of the auricle. I again cannot admit this as evidence, because chronic distension of any viscus is always associated with hypertrophy of all its coats.

For the sake of argument let us assume that the auricle as a whole does contract so as to expel its contents with some force into the ventricle. We must not lose sight of the fact that the venæ cavæ and hepatic veins have no valves, and that the valves that are found at the junction of the internal jugular with the subclavian and subclavian with the external jugular are only competent during moderate distension. The sinuses of the head and great veins can be injected from the superior cava. With these facts before us, it is impossible to deny that were the auricle as a whole to contract with any degree of force, a back flow and distension, with pulsation of the great veins at the root of the neck and liver, must inevitably result. The auricle would exert itself quite as much in one direction as the other; in fact more so, for the combined openings into the cavæ are larger than the opening into the ventricle, and the opposition is *less*. What takes place when there is back pressure into the auricle from valvular incompetence of the right heart? Why, frequently a

well-marked venous distension and pulsation, not only in the neck, but in the liver also. It is to my mind impossible to argue that the momentum of the flow in the veins and liver normally arrest a back current and prevents pulsation. If this is the case normally, why not during tricuspid regurgitation?

I think, then, I have adduced sufficient evidence to support the argument that the auricle, as a whole, does not contract more than perhaps to a most limited extent.

To sum up: For the contraction I know nothing. Against, (1) thinness of walls or inability; (2) absence of valves in superior or inferior cavæ; (3) no evidence of endo-ventricular or endo-auricular pressure on pressure curve; (4) auricular systole not necessary.

According to this view, the auricular appendix being the only portion of the auricle that vigorously contracts, the question now arises, How is it that the auricular appendix can contract and discharge its contents without affecting the general tension in the auricle? The explanation is to be found in what one may any day observe in a marine aquarium. The common medusa, for the purpose of locomotion, discharges its contents with considerable energy. The reaction of the discharge causes the medusa to progress with more or less rapidity, whilst the contents are moved with considerable rapidity in the opposite direction without disturbing the surrounding fluid. As a further illustration, take an ordinary brass syringe as used by aurists; fill this with coloured fluid, and lower it in a bath of clear water. On rapidly discharging the contents of the syringe it will be seen to pass in a stream without materially affecting or mixing with the surrounding fluid. The Gulf Stream is another familiar example. The auricular appendix discharges its contents directly into the ventricle with considerable rapidity without imparting an increase of tension to the auricle* (see diagram, page 10).

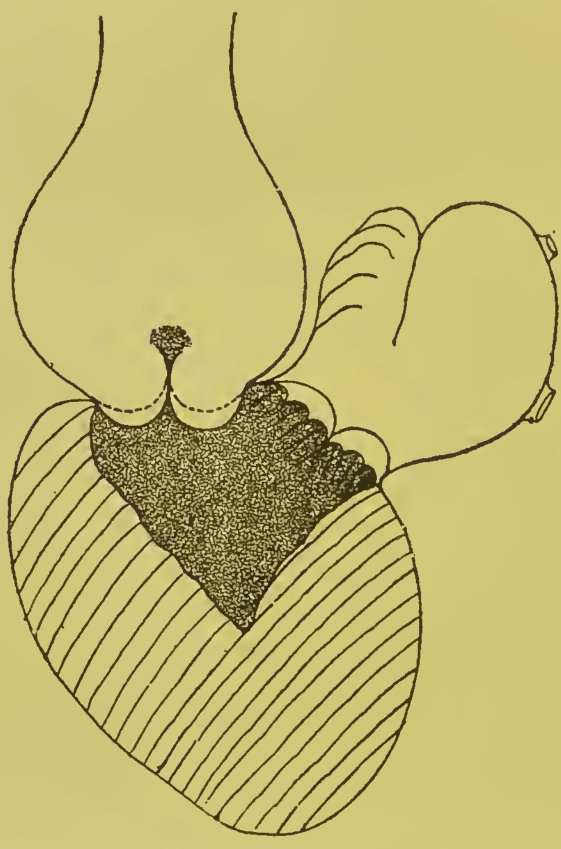
* This would only be possible where the pressure is low.

The next question presenting itself is the effect on the ventricle or its contents by this rapid discharge from the auricular appendix. The object of the rapid discharge from the auricular appendix, I think, will become apparent when studied side by side with the closure of the tricuspid and mitral valves. These valves differ considerably in their mode of action from the semilunar valves. Taking the tricuspid orifice, the valves may for all practical purposes be compared to three parachutes, only capable of closing an orifice when much distended; the more the distension the more efficient the closure. From this it will be seen that in order to bring these parachutes into requisition they must first be opened out by the contraction of the musculi papillares. As the ventricle becomes dilated and filled the valves will become distended, but not sufficiently distended to prevent a reflux into the auricle. Directly the tension begins to rise in the ventricle the valves will become distended and closely approximated. The first impulse to the rise of tension in the ventricle is given by the rapid addition of the contents of the appendix to the ventricle. This rapid addition of blood will have most effect, as previously explained, on the valves, or, in other words, where the ventricle is thinnest. I submit to you that the auricular appendices, by discharging their contents suddenly and rapidly into the ventricle, complete the necessary distension of the parachutes of the valves so as to efficiently close the auriculo-ventricular orifices preparatory to the contraction of the ventricles.

From what you may have gathered from my views as to the action of the valves and their mechanism in closing the auriculo-ventricular and aortic openings you will not be surprised to hear that I cannot accept the current explanations of the sounds that are a prominent feature of the normal heart beat.

We are all aware that on listening to most mammalian hearts with the stethoscope or naked ear applied to the chest wall two

distinct sounds are heard synchronous with each cardiac revolution. The two sounds differ from each other in quality and point of time, and are undoubtedly due to distinct causes, though



Dotted lines indicate Position of Valves before Ventricle commences to relax. Thick line gives position of Valves on relaxation of Ventricle. Towards end of Systole last drops of blood entered and *in situ*. Valves closed.

one is capable of being affected by the other. That the sounds originate in the heart, and are in some way produced or affected by the passage of blood through the heart, we are all agreed. The widest difference of opinion exists as to the exact share or

what share the blood valves and muscular tissue take in the formation of these two sounds. Any detailed description of these two sounds from me is unnecessary. Suffice it to say that the first is prolonged, low-pitched, and booming, and the second, short, sharp and high-pitched. The first sound is synchronous with the systole of the ventricle, and the second with the commencement of the diastole. On examining the various works on physiology there seems to be a complete agreement as to the cause of the second sound. The books say, "There can be no doubt, in fact, that the second sound of the heart is due to the semilunar valves being thrown into vibration at their sudden closure."

Now, I for one beg leave most respectfully to differ from the authors of this statement. If they will allow the quotation to run thus, I am in perfect accord with them: There can be no doubt, in fact, that the second sound of the heart is due to the semilunar valves being thrown into vibration subsequent to their closure, and consequent on the loss of support below, due to the relaxation of the ventricle.

I will now proceed to justify the recommendation of this statement in preference to the quotation laid before you.

I have pointed out in a previous lecture how as the ventricle discharges itself the tension in the first part of the aorta increases, exerting an increased pressure on the semilunar valves and an increased opposition to the entrance of blood from the ventricle; that consequently the last drops of blood that enter the aorta must do so under very great pressure and with very small momentum; that at this precise moment the tension above and below the valves is approaching equality, and at the very moment when the tension above is by ever so small an amount greater than that in the ventricle the valves must close to. In fact, the closure must of necessity immediately follow the entrance of the last drops of blood into the aorta, or

regurgitation would inevitably take place. At the precise moment when the last drop of blood has entered the aorta the pressure above the valves and below is equal and at a maximum. The ventricle now immediately commences to dilate, and the tension below the valves is suddenly changed from a maxi mum to a minimum. The valves now have to bear the whole strain of the aortic pressure without the smallest assistance from below. This strain is not gradual, but sudden, from the sudden relaxation of the ventricle, the valves being subjected to a sudden extra strain at the moment of commencing diastole of the ventricle vibrate. The vibration is short, sharp, and high-pitched the duration is unmeasurable. The second sound of the heart may be exactly imitated by suddenly stretching a small portion of an ordinary silk handkerchief between the thumb and forefinger of each hand.

I beg to submit to you that the second sound of the heart is due to this sudden change of pressure in the ventricle. The support below the valves being suddenly changed from a maximum to a minimum, they are strained, and being consequently ren dered suddenly more tense, vibrate. The second sound is a sound of tension, a sound of stifled or aborted vibration.

In dealing with the first sound of the heart there is even more difference of opinion as to its exact nature and causes. It is more prolonged, lower pitched, and booming than the second sound. It is synchronous with the contraction of the ventricle, commencing shortly after the commencement of the ventricular systole and ending with the systole. It is distinctly affected by the degree of vigour or the reverse of the ventricle; hypertrophy intensifies, whilst atrophy or degeneration distinctly lessens the sound. It is generally attributed to two causes: firstly, to the sound emitted by the contracting fibres of the ventricle (even this is disputed); and secondly, to the sound produced by the

auriculo-ventricular valves and cordæ tendinæ being thrown into vibration at the moment of their sudden approximation.

With regard to the first factor, namely, the sound produced by the contracting fibres of the ventricle, it is impossible to reject it in the face of the fact that the sound is emitted from excised and empty hearts whose valves are destroyed or fixed. I believe there is very little disagreement on this point.

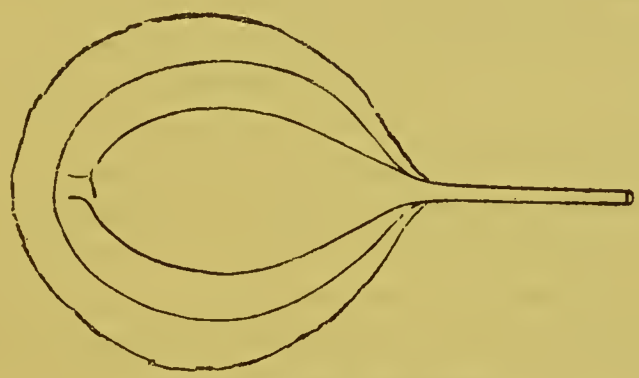
In reference to the second factor, namely, the sound produced by the sudden closure and vibration of the auriculo-ventricular valves and chordæ tendinæ, I am unable to agree. I have done my best to place before you very cogent reasons why I consider it impossible to entertain the notion of a sudden or slap-bang closure of the parachutes. To my mind it is impossible to conceive how the openings into the auricles can be closed by structures like the valves, except in the manner I have related to you. Again, I submit to you that neither the semilunar valves nor the auriculo-ventricular valves are approximated suddenly in the sense the books would imply. At both orifices the closed valves are subjected to a sudden strain. In the case of the semilunar valves the sudden strain is due to the loss of support by the ventricle; and in the case of the auriculo-ventricular valves the strain is due to the sudden and powerful contraction of the ventricle. The sound is a sound of tension or muffled vibration, not of free vibration.

To repeat more exactly, the first sound of the heart is due to the sound emitted by the contraction of the muscular fibres of the ventricle, as well as the sound emitted by the sudden strain that the closed parachutes are subjected to during the rapid contraction of the ventricle. Possibly another element may be the sudden strain the aorta is subjected to during the systole of the ventricle.

LECTURE III.

MR. PRESIDENT and Gentlemen,—In my last Lecture I endeavoured to lay before you my views on the part played in the mechanism of the circulation by the sinuses of Valsalva and the auricular appendices. I passed then on to discuss, first, the second sound of the heart; and here I differed from all previous observers, in so far as I contended that the semilunar valves did not close too suddenly in the sense the books would imply. I contended that the valves must of necessity be nearly closed before the last few drops of blood entered the aorta; that at the moment when the last drops of blood had entered the aorta the pressure above and below the valves was equal, in fact the valves were suspended between two equal and opposing forces; that as soon as the pressure above exceeded the pressure below no more blood could enter, and the valves must close to immediately after the entrance of the last drops of blood. I attempted to show that any other arrangement than this must of necessity allow regurgitation into the ventricle. I attributed the second sound of the heart to the strain the closed valves were subjected to, consequent on the sudden loss of support below, at the moment of commencing diastole of the ventricle. In like manner I endeavoured to show that the tricuspid and mitral valves could not flap to suddenly. Any such arrangement would undoubtedly imply regurgitation as a normal state of things and that there was not the smallest particle of evidence in support of this. I suggested that the valves were gradually

approximated by the musculi papillares, from the very commencement of the diastole of the ventricle; that they could only become efficient as valves when much distended, and that their efficiency and approximation depended upon the distension of the ventricle; also that the complete distension of the ventricle and consequent approximation of the valves depended on the sudden discharge from the auricular appendix. Lastly, I contended that the tricuspid and mitral valves being closed, they were suddenly



Elastic Ball with Rigid Tube.

strained by the powerful contraction of the ventricle. Being strained, they probably emitted a muffled sound of tension, and that this, probably with the sound emitted from contraction of the muscular tissue of the ventricle and the sudden tension of the aorta, was the cause of the first sound of the heart.

Before proceeding to discuss the mechanism of the pulse, and whilst on the subject of the contraction of the ventricle and cause of the second sound of the heart, I would here wish to fill up some omissions in my last Lecture.

I would have added that I do not see how it is physically possible for the ventricle to completely empty itself. There is always an undischarged residuum, as I think will appear evident on a further examination of this endo-ventricular pressure tracing. The ascent from E to F (see diagram, page 4) is caused by and indicates the initial rise of pressure in the ventricle due to the contraction of the auricular appendix. The subsequent slight fall is due to the bulging and approximation of the tricuspid and mitral valves towards the auricular cavity. The almost vertical rise from G to B represents the continuous steady and powerful contraction of the ventricle, the point B marking the phase of maximum tension both in the ventricle and the aorta, and the exact point when the last drops of blood have entered. The more or less sudden descent from B to C, and subsequently to D, causing a well-marked elevation in all tracings, is the subject of grave difference of opinion.

Mr. Rolleston says, "With regard to the cause of the appearance of this notch, only one explanation of which I have been able to think appears to me satisfactory, . . . it may be due to the increased freedom of outflow from the ventricle which takes place as soon as the column of blood is set in motion."

The explanation I have offered for your acceptance of the cause of the second sound of the heart will satisfactorily explain this notch, and subsequent slight rise or more gradual fall to D. The valves at the moment of commencing diastole were suspended, but closed between two equal and opposing forces. The support below being suddenly removed, the valves are forced down towards the ventricle, and for a moment the displacement serves to slightly increase the tension in the undischarged residuum of the ventricle, causing the slight elevation at the point D. At D, then, is the point where the second sound of the heart is produced. The subsequent rapid and continuous fall to the point E marks the aspiration of the auricle and great veins by the ventricle.

We pass on now to examine and discuss the mechanism of the pulse.

If asked to define the pulse, or to explain the cause of the something we feel on applying the finger to any prominent artery on the living subject, I should say that the feeling of enlargement of the artery imparted to the finger at the point of contact was an actual temporary enlargement of the artery, due to a wave of distension propagated from the aorta. I state simple facts.

I made experiments on the arteries of dogs from the aorta downwards. I placed the finest aluminium calipers on the aorta of several dogs, exerting no sensible pressure, each limb of the calipers passing round an arc of the artery equal to a semicircle. The calipers were always markedly separated, as indicated by the recording points. The same experiment on any artery gave exactly the same results, and with calipers placed simultaneously on the aorta and femorals the femoral dilatation was subsequent to that in the aorta.

I hope this is a sufficient answer to the statement that the pulse as felt at the wrist or elsewhere gave one the impression of distension; that really no distension took place at the point of contact; the artery simply returned to the cylindrical shape, in opposition to the flattening caused by the pressure from the finger or sphygmograph.

I would maintain, then, that the pulse is caused by a wave of distension propagated from the aorta, or, in other words, the distension imparted to the aorta by the ventricular systole is distributed rapidly throughout the arterial system. The first part of the aorta being over-distended by the ventricle, recoils upon its contents. The passage into the ventricle being closed, and the tension or pressure on the upper surfaces of the semilunar valves being at a maximum at the moment of commencing diastole, the recoil of the aorta can have no further effect upon them.

Such being the case, part of the force of recoil is expended in dilating another segment of the tube, whilst the aorta retains for itself an increased tension, which increased tension only subsides to the normal before the next systole of the ventricle. The gain of tension in the aorta after the recoil serves to squeeze the blood through, and oppose the opposition to the flow in the capillaries. The next segment being over-distended, recoils, and whilst reserving in like manner an increase of tension for itself, having a like object, distends a segment of the tube farther removed from

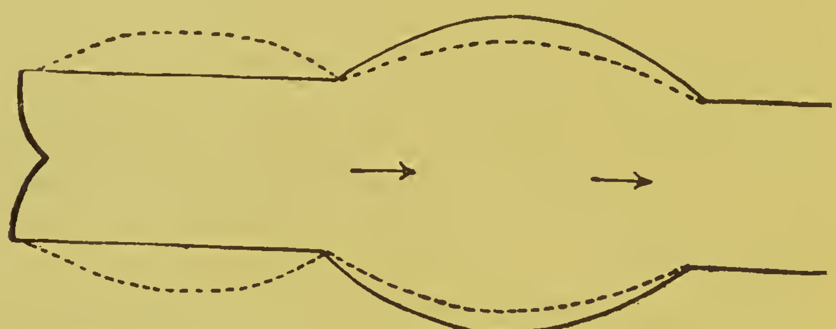
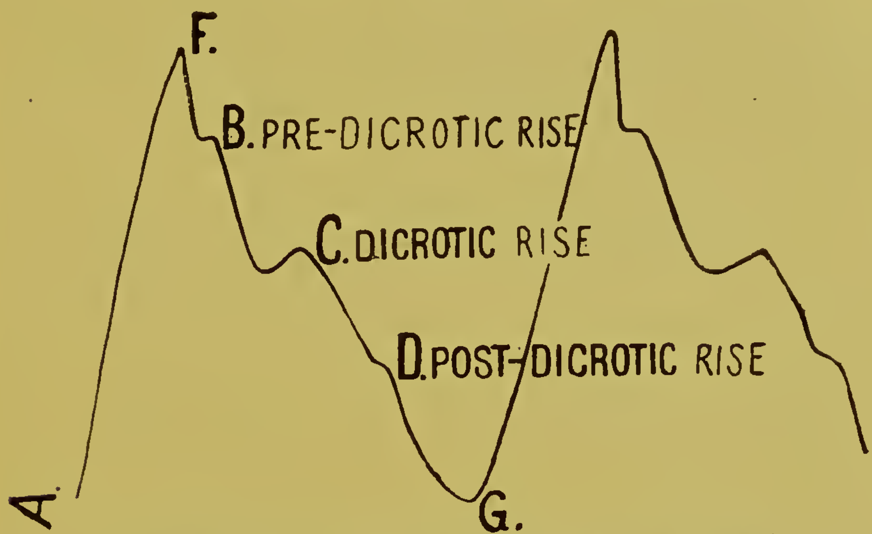


Diagram indicating passage of Wave of Distension. Segment after Segment distended and recoiling.

the heart. The recoil of the second segment of the tube normally has no back effect on the portion of the aorta nearest the heart, because here the walls are thicker and the tension is higher, and because, moreover, the resistance to distension in front is less than behind. The semilunar valves practically serve the same purpose, when the tension is normal, as if they followed the wave of distension, backing up, as it were, each segment of the tube, and compelling this last segment to dilate a segment farther removed from the heart. The rate of distension is calculated to travel with the rapidity of about two metres per second. We

thus see how it is that the initial distension of the first part of the aorta by the ventricle is rapidly distributed throughout the arterial system, in order to maintain a general state of tension throughout the arterial system, so necessary for the circulation of the blood.

We thus see how it is that the tension in the aorta nearest the heart is the highest, and see how the associated tension and dis-



NORMAL PULSE TRACING. RADIAL (MAN)

tension gradually subsides towards the capillaries. Thus much is simple, and will be disputed by few.

Not being satisfied with these simple broad facts about the pulse, various observers have endeavoured to measure or record, by means of various instruments, the movements of the wall of the artery at a particular point during its temporary distension and collapse. The instruments in question are the various forms of the sphygmograph, so familiar to us all.

I do not propose to go into the history of the sphymograph, or

describe its mode of application or forms. This would be quite foreign to my purpose and quite unnecessary. All I propose to do is to take any normal average tracing, say from the radial artery of a healthy man, and examine its purport, and interpret, if possible, its meaning (see diagram, page 43).

A tracing such as is represented by the diagram is a fair specimen of a normal pulse tracing taken from the radial artery of an apparently healthy man. In the first place, it is admitted by every observer that the curve as represented by the zigzag line from A to G does represent with more or less accuracy the temporary distension and collapse of that portion of the arterial wall in contact with the button of the lever. The line of ascent from A to F is, moreover, called the anacrotic elevation, and the line from F to G the katacrotic fall. Any elevations or notches occurring on the line F to A are, moreover, termed anacrotic, and those on the descending are termed katacrotic. A normal tracing usually shows no anacrotic notches, but usually three or four notches and corresponding elevations are always apparent on the katacrotic line. These are marked in the diagram B C D. Under certain circumstances, which we shall investigate more fully presently, the finger when placed on the living artery is capable of appreciating two distinct impulses to each beat of the heart. A pulse of this character is called double pulse, or dicrotic. If now a sphygmograph be applied to the artery in question a tracing such as that represented in the diagram is readily obtainable.

The peculiarity of this tracing is that the elevations B and D in the normal tracings are suppressed, but the one marked C is greatly exaggerated. On this account, and for these reasons, it is customary to name the elevation C, in the normal tracing, the dicrotic rise or elevation. Moreover, any elevations occurring on the katacrotic line between A and C are spoken of as pre-dicrotic, and

any between C and G as post-dicrotic. It is more advisable, I think, to retain these simple terms, in preference to using the terms percussion and tidal waves and secondary tidal waves, as thereby we assume a cause for such elevations which, to my mind is incorrect.

Thus far, then, I have simply placed before you a pulse tracing, and indicated the terms I think it advisable to adopt. The next point is the interpretation of these elevations on the katacrotic line, and more especially I shall direct your attention to the one marked C, or dicrotic elevation, which is liable under certain circumstances to be greatly exaggerated (as in diagram).

DICROTIC PULSE.

Case of Typhoid fever.

1 2

Assuming proportions almost equal to the initial rise. More ingenuity has been expended in devising theories for the interpretation of these elevations on the katacrotic line than any other subject in the whole realm of physiology. I do not intend to inflict upon you these theories or their refutations; I simply propose to lay before you the explanations that, at the present day, find most favour with physiologists. The honours seem to be

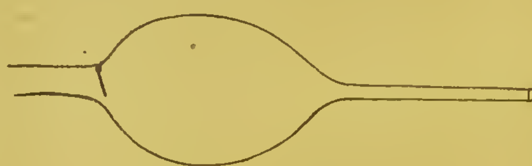
equally shared by Dr. Galabin and Professor Burdon Sanderson whose names I mention to you with the very highest possible respect. To quote from either of these authors would be too lengthy, so I have turned to the last edition, dated 1887, of the well-known work of my much respected teacher and friend Dr. Bristowe. I think we have a right to assume that the work in question contains the latest and most acceptable views on the subject. What are these views ? I will quote from the work :—

"The typically complete tracing would thus present not less than four successive waves, of which at least two would correspond to the systole of the heart, and at least two to the diastole The first of these waves, which is known as the primary or percussion wave, is generally attributed, not to any actual addition to the quantity of blood which the artery presenting it already contains, but to the impulse which is supposed to be transmitted along that blood by the shock of the commencing systole, and which is supposed to precede by a scarcely appreciable interval the secondary or tidal wave which follows it. Dr. Galabin, however, shows that this explanation is incorrect, and that the percussion and tidal waves form in the artery but one wave, and are only separated by the sphygmograph. Owing, he says, to the inertia of the long lever, it is carried up a little too high, and when in falling it meets the true arterial wave it is again tossed up, and thus forms the tidal wave. The third, or dicrotic wave has, like the first, been variously explained. It has been attributed by many to the shock of the sudden closure of the aortic valves, an opinion in which Dr. Galabin concurs, and again to the recoil of the hitherto distended arteries. But the cause is probably that which Dr. Sanderson assigns to it. He points out that as the wave due to the injection of the ventricular contents into the aorta takes a certain time to reach the capillaries, and as hence, the period of greatest movement in the latter vessels must

take place distinctly later than in the aorta, so the subsidence of this wave and the period of comparative rest that marks the end of the systole and the whole of the diastole is likewise delayed in transmission to the peripheral vessels, and that, consequently, there is a moment at which, while the blood is almost stagnant in the aorta, it is still flowing rapidly in the minuter vessels, and a later period at which the blood in the capillaries also becomes comparatively quiescent. But this arrest in the capillaries, accompanied as it is by the recoil of the elastic arterial coat upon the diminished contents of the vessels, produces a virtual distension and a sudden increase of pressure throughout the arterial system. The dicrotic wave is the expression of this arterial tension. The fourth wave has probably, as Dr. Galabin considers, the same relation to the dicrotic wave as the tidal to the percussion wave."

Now, I need not tell those of you who have done me the honour to listen to my first and second lectures that I am unable to accept these explanations. I could give you, if I had time, my reasons, but in preference I will lay before you an explanation at once, I trust, simple and in accordance with facts. I do not think we need import into the matter any elaborate arguments or theories. Starting from the point of distension of the first part of the aorta and its recoil, I pointed out how the first part of the aorta was distended by the ventricular contents; how this distension was distributed with the rapidity of over two metres to the second throughout the arterial system; how each segment, when distended, retained for itself a portion of its addition and passed the surplus on, and so on to the smallest artery; how also by this means the general tension throughout the arterial system was maintained, thereby maintaining a constant and equable flow in the capillaries, and allowing no periods of stagnation as stated by Dr. Sanderson.

I would here ask your attention to this simple diagram, as very diagramatically representing a portion of an artery along which the wave of distension is passing. This simple indiarubber ball in like manner may be taken to represent the portion of the artery enlarged by the wave of distension. Why the wave of



distension continues to travel in one direction from the heart to the capillaries I have explained to you: (1) Because the tension in the proximal portion of the artery is considerably higher than the tension in the distal portion. (2) Because the blood cannot move towards the ventricle, and it can towards the capillaries, and is moving. (3) Because the wall of the artery of the penultimate portion is thicker than the wall of the artery in front of the wave, and resists, normally, further distension.

It is perfectly clear to any one that in the absence of one or more of those three causes there is no earthly reason why on the recoil of any distended segment of the arterial system the portion of artery in front of and behind the wave should not be equally distended, just the same as in this simple indiarubber ball, if no valve exist at this point as much of its contents will pass one way as the other; or, to make our illustration more complete, the recoil of this bag will affect and dilate the tube of entrance as well as the tube of exit. It matters not whether a wave be considered as passing, and liquid flowing through the bag, or whether the contents be stationary. Practically speaking,

the effect of the semilunar valve follows the wave of distension, and performs for each segment of the arterial system the same function as it did for the first part of the aorta.

Now, sir, it is impossible, to my mind, to conceive how the recoil of the segment under consideration can take place without producing some effect, however slight, on the penultimate segments or the portions of the artery last distended. On recoil of the segment under consideration, the greater part will be expended in dilating a segment of the tube farther removed from the heart. A small portion will be expended in slightly redistending the penultimate portion. This penultimate segment will again recoil and distend the ultimate portion, and so on as long as the tension in the artery and duration of the diastole will permit. High tension in the arterial system would minimise or annul these slight redistensions; low tension would increase and exaggerate them. Let us look at the facts of the case.

Pulse of vigorous health. Tension high.

Contracted granular kidney Hypertrophy Left ventricle

Arterial tension of chronic tubal nephritis

Here is a tracing taken from a case of chronic renal disease, with hypertrophy of heart, where the tension is always high. The secondary elevations have almost disappeared. Here is a tracing

taken from a case of typhoid fever, where the secondary elevation is almost equal to the primary (see diagram, page 45). The vessel is so lax, and the heart is beating so quickly, that only one of the secondary elevations can be recorded. Just as in this indiarubber ball,* if the valves be removed to some distance, I defy you to arrange matters such, however tense you like to make this portion of the tube, however free the exit, and however thick the wall within the limits of rigidity, without, on a sudden contraction of the ball, producing some dilatation of this portion of the tube. Of course the tenser the tube *pro rata* the less effect.

Gentlemen, I hold that the slight elevations on the katacrotic line, as evidenced by the sphygmograph, are due to this cause. I submit to you that the above is the correct explanation of the dicrotic and other elevations on the katacrotic line. To repeat more exactly, I hold that the slight elevations, as seen on the katacrotic line, as evidenced by the sphygmograph, are due to the slight redistensions that must inevitably take place in the penultimate segment of artery on the recoil of the part; that this penultimate segment under examination reacts on the ultimate, and the ultimate on the penultimate, as often as the tension and the diastole of the ventricle will permit.

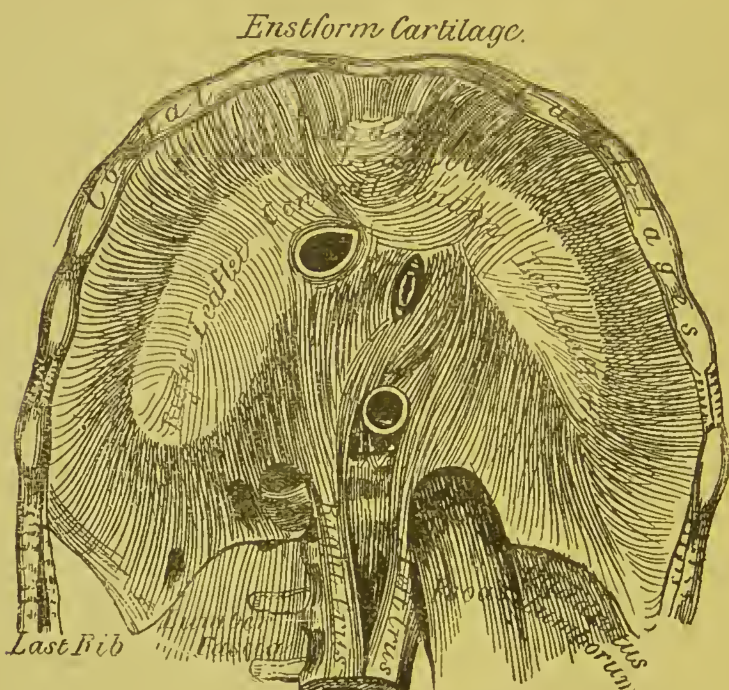
Before bringing these lectures to a close, in the short space of time that remains to me, I would wish to direct your attention to certain peculiarities in the portal circulation, and make a suggestion. The peculiarity of the portal circulation is this, that whereas the arteries come off from the systemic trunks, the veins do not return immediately into the inferior cava, but are collected by a great vein, termed the portal. This great vein dilates into a sinus in the portal fissure of the liver, and from thence breaks up into a system of capillary plexuses offering an

* Here was shown an ordinary indiarubber syphon enema syringe.

enormous resistance to the onward flow of the blood. The splenic artery passes into the enormous swamp, as it were, of the spleen, and any *vis a tergo* derived from the ventricular systole must be fully expended in this organ. The tension in the portal vein is stated on the best authority to seldom exceed a few mm. of mercury. In my experiment the manometer fell from +4 mm. to −2 mm. on connecting it with the interior of the splenic vein. I do no pretend to be able to speak authoritatively on the subject. The broad question, however, presents itself, By what means is the enormous opposition to the flow of blood in the capillaries of the liver overcome? The systole of the ventricle takes no part, as evidenced by the low tension in the portal vein. The possible agents are—(1) the rhythmical compression of the abdominal contents during respiration; (2) the aspiration of the thorax; (3) the aspiration of the right ventricle; (4)—the last and least likely—the hepatic artery.

I believe all these agents take part in maintaining the portal circulation. The greatest share I would assign to the diaphragm as the chief agent in compressing the abdominal contents. The whole of the posterior and upper surfaces of the liver are in contact with the under surface of the diaphragm. The diaphragm grasps the liver to the extent of more than a semicircle. In the same way the whole of the large outer surface of the spleen is in contact with the arched fibres of the diaphragm. The diaphragm grasps the whole of this outer surface. The respiratory function of the diaphragm has, I think, been much exaggerated. The central tendon of the diaphragm is fixed, by reason of its attachment to the pericardium and the passage of the two large tubes, the œsophagus and the vena cava. In like manner the lower attachments of the diaphragm are fixed from tip of last rib to tip of last rib. The ligamenta arcuata, interna and externa, are immovable. Between the central

tendon and the ligaments in question the muscular fibres arch downwards in a vertical direction. The result of contraction of this portion of the diaphragm would be to straighten the curve, and protrude the liver and spleen towards the epigastrium, at the same time compressing them. In like manner, if the lateral portions of the muscle contract between two fixed points the arch is



Diaphragm (after Gray).

straightened and the abdominal contents are compressed. The antagonism of the intercostal muscles, raising and dilating the lower ribs, prevents their being drawn in by the diaphragm during ordinary respiration. Two methods of inspiration are known to

physiologists and teachers of singing—the one where the epigastrium is hollow, the diaphragm pushed up to the fullest extent, and the lower ribs much raised and dilated; the other where the diaphragm is lowered to the fullest extent, and consequently the abdomen protruded at the epigastrium. The first is the method of inspiration taught by the old Italian masters, the second by the new school. Which method affords most breathing capacity and control? Why, the first, I would suggest. A glance at this formula will convince you: *$V = \frac{h}{3} \pi r^2$. However large h is, it is always divided by 3. It can never be large at any time. However small the addition to r, it is squared. Seeing, then, that the diaphragm cannot descend far, on account of the fixation of its central tendon, and that if it could descend it is not so efficient as an inspirating agent as if it ascended to its fullest extent, I think I was justified in merely suggesting that the diaphragm as an inspiratory agent was overrated, but by compressing the abdominal contents materially assisted in the circulation of the blood through the liver.

In conclusion, I have to thank you, sir, for your patience under what I cannot but conceive must have been to you an infliction; and you, gentlemen, for your patient hearing and forbearance with my many deficiencies.

* If V = volume of cone, h the height, and r the radius of base, and π the constant relation of the diameter of a circle to its circumference, then $V = \frac{h}{3} \pi r^2$. The chest cavity practically represents a cone.

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